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Acne in Mature Members of the Community

BY

J. A. W. MILLER-ORANKO,

M.B., CH.B.(CAPE), D.P.H.(LONDON), DIP.DERM.(LONDON)
Dermatologist, Salisbury

Acne is so common a complaint that junior school children know that some of them will develop spots and pimples as they grow up and that as they mature they will grow out of them. Their doctors will tell their parents that pimples are due to hormones and that they are part of the growing up phase. Some doctors will state that acne is condensed into a ten-year period, and that the child with spots at the age of 12 should be clear-skinned before the age of 22. Acne cannot be prevented, neither can it be cured, but it can be controlled. It has, therefore, provided a wonderful field for quack cures, old wives' tales and for cosmetic remedies amongst teenagers.

However, we see patients in a mature age group who are shy and embarrassed by their teenage-type spots who pluck up courage to get medical advice. Table I refers to 20 such patients who have presented over the past 14 months. These figures are interesting in that they illustrate the fact that acne vulgaris is common amongst women, most of whom are mothers with children and who are in their late second and third decades. It would be unwise to draw any statistical conclusions from so small a survey, but it is worthy of note that nine, or roughly half, of them have never had acne before and that six of them associate the onset of their condition with their confinement. Eighteen of them are married and 18 of them enjoy good health. One patient had red hair, six were blond, and the remainder either had light brown hair or were frankly brunette. The distribution of the lesions has been in recognised acne areas in all cases, and mostly facial. Pre-menstrual exacerbation was complained of by six of them. The average age for the group was 32 years.

We know that the condition "seborrhoea" occurs when the production of sebum is excessive

for the age and sex of the patient. It is often genetic in origin, and on going into the family history other cases are often revealed. Seborrhoea can be a factor in certain neurological conditions such as Parkinson's syndrome, post-encephalitic Parkinson's especially, epileptics and in some manic depressives.

Comedones (blackheads) are black or dirty-brown horny plugs found in the pilosebaceous orifices. They can be expressed by squeezing and for this a comedo expressor is recommended. Comedones are found in—

- acne vulgaris;
- familial comedones — due to an autosomal dominant and new comedones can continue to develop into middle age;
- acne venenata — due to an exogenous agent following therapeutic irradiation of skin tumours;
- pseudo xanthoma elasticum;
- solar elastosis.

Acne itself is a chronic inflammatory disorder of the pilosebaceous follicle and Rook, Wilkinson and Ebling use the following classification:

- Acne vulgaris.
- Acne neonatorum.
- Acne of external chemical origin (acne venenata).
- Acne medicamentosa.
- Acne conglobata.

The age for acne vulgaris is variable and normally facial lesions begin between the tenth and thirteenth years, reaching the greatest severity between the fourteenth and nineteenth years. Rook *et al.* state that the severity and incidence decline steadily during the third decade, but some active lesions are present in over 10 per cent. of adults aged 30-40 and exceptionally may continue into old age. This is an important statement and revealing to many. However, acne normally affects young people and clears up soon after 20 years of age and it may clear on marriage or after pregnancy.

As stated earlier, acne is a disease of the pilosebaceous apparatus and is characterised by the formation of a comedo (blackhead) which is due to an inspissated plug of keratinous debris in the pilosebaceous duct. We know that acne occurs with a higher testosterone level and that pubescence is the time of acne and that it disappears with maturity. We know that the sebaceous glands secrete a sebum that is qualitatively normal but quantitatively abnormal.

In the simple pustules of acne we do not find normally pathogenic bacteria, but we do find *propionibacterium acnes* and *staphylococcus albus*. There is a theory that the aetiology of acne is closely tied up with the obligate anaerobe *propionibacterium acnes* that digests fat and splits it into fatty acids as far as propionic acid, which is a highly irritating fatty acid, and when produced this gives rise to inflammation.

We know that the simplest basic function of an epithelial cell is the manufacture of keratin, and thus when a sebaceous gland has its outlet blocked the glandular epithelium changes over to the manufacture of keratin and the gland then appears like a little white cyst. These cysts are commonplace on the faces of chronic acne sufferers.

Some of these cysts may have the bacillus *propionibacterium acnes* left behind within them. This bacillus will then convert the sebum into propionic acid, and if some of this irritating acid leaks out a chemical fatty inflammation will ensue. It is around this fatty inflammation that white blood corpuscles will collect, forming a sterile pus which is the classical pustule of acne.

In case this explanation is a simplification of the matter, we must consider some of the articles in current literature, some of which support and some of which are at variance with this theory.

Kligman and Katz state that supporting evidence that human scalp sebum contains substances that directly stimulate the follicle to form a comedo was obtained by daily applications for ten days of human scalp sebum to the external ear canal of the rabbit. They accept that the formation of the comedo is the key question in the pathogenesis of acne and use the following circumstantial evidence that acne vulgaris is dependent on the activity of the sebaceous gland:

- (1) Pre-pubertal children with their tiny sebaceous glands do not experience acne.
- (2) Oestrogens abort the disease by suppressing sebum production.
- (3) Shrinkage of the sebaceous glands by x-rays is an effective, though temporary therapeutic measure.
- (4) The severity of acne vulgaris is in direct proportion to sebum production.

In discussing the fact that sebums of all individuals are comedogenic, the authors conclude their article by stating, "sebum appears to be a necessary but not a sufficient condition in the multifaceted aetiology of acne. Sebum may fuel the process, but not everyone catches fire. Individual susceptibility is decisive but, unfortunately, inexplicable." Kligman then goes on in a further article to show how the comedogenic potency of sebum is considerably less than that of the chemical *acneigen* Halowax. The comedones produced in a rabbit's ear from sebum are smaller and never attain maturity.

The question of androgens in acne is discussed by J. S. Strauss and P. E. Pochi. These authors show how the active hormone progesterone is also a precursor for aldosterone, cortisol, testosterone and oestrogens. They refer to the normal levels of plasma testosterone in the male as ranging from 0.55 mu.g./100 ml. - 0.8 mu.g./100 ml. of plasma;

and in the female as ranging from 0.33 $\mu\text{g.}/100$ ml. - 0.18 $\mu\text{g.}/100$ ml. of plasma. In pre-pubertal boys and girls, plasma levels of testosterone are low. A diurnal pattern of testosterone levels has been found in males but not in females, though females have a slight variation in plasma testosterone in relation to the menstrual period. There is a slight inconsistent rise in the luteal phase of the menstrual cycle. The production rate for testosterone as determined by the urinary measurement is 6-8 mg. per day in men and 1-2 mg. per day in women.

It is generally agreed that in the male testosterone is produced mainly by the testicles. There is evidence that the ovary secretes androgens in the females and we know that the human adrenal cortex synthesizes testosterone.

It is now considered that the skin may be one of the sites for the metabolism of androgens. The authors discuss the condition called testicular feminisation, where it is assumed that there is an inability of the target tissues to respond to androgen. The sebaceous glands show low sebum production and thus the sebaceous glands show an androgen unresponsiveness together with other androgen target tissue.

In the polycystic ovary syndrome, sebum production is higher in normal women in the same age group and the sebaceous glands are presumably stimulated by the increased androgens in this disease.

These authors discuss acne as a hormonal disorder and state that plasma and urine testosterone levels are normal in acne vulgaris, and that females with acne are not more virile than normal unless there is an obvious ovarian tumour or Cushing's syndrome, etc., and they state that it is theoretically possible that there may be altered metabolic transformation of the androgens within the sebaceous gland itself to explain the seborrhoea that most patients with acne manifest.

J. A. Milne refers to the work of Bailie *et al.* (1966), who investigated various hydroxysteroid dehydrogenases in human sebaceous glands and confirmed that histochemical evidence of these enzymes was confined to the anatomical areas prone to develop acne vulgaris; and further, the work of Cameron *et al.* (1966), who reported the formation of small amounts of testosterone from dehydroepiandrosterone in increased amounts by skin from an acne area compared with a non-acne area having the same numbers of sebaceous glands per unit area. He feels that there is sufficient evidence to implicate the sebaceous gland solely as the major source of enzyme activity, though there is as yet no absolute proof of the central role of

the sebaceous gland in steroid biosynthesis. There is an increased steroid metabolic turnover in the acne areas, and this increased androgen conversion may be responsible for the clinical distribution of acne vulgaris. Dehydroepiandrosterone disappears shortly after birth and re-appears in the teenage period and may be responsible for the onset of acne in the puberty period.

In discussing seborrhoea one cannot overlook the recent article by J. L. Burton, W. J. Cunliffe and Sam Schuster. These authors refer to the work of Krant *et al.* (1968), who found that patients with advanced breast cancer had seborrhoea — they therefore compared the sebum excretion rates in patients with early breast cancer and a matched group of control subjects. They agreed with Krant *et al.* (1968) that the rate of sebum excretion increased in patients with breast cancer. They state that this seborrhoea could have been of recent onset in patients with breast cancer, or it may have been present for many years and that the possible causes of the seborrhoea could be—

- (1) increased levels of circulating tissue sebotrophic hormones;
- (2) an enhanced end organ response to such hormones;
- (3) decreased inhibition of sebaceous gland activity by
 - (a) decreased levels of circulating or tissue inhibitors, e.g. oestrogen; or
 - (b) diminished end organ response to inhibition.

The role of fatty acids in acne vulgaris has also been the object of much research. R. E. Kellum* in discussing free fatty acids from C_2 - C_{16} states that though pathogenic factors have not yet been defined in acne vulgaris, there is indirect evidence that free fatty acids play a role in the pathogenicity of acne vulgaris, and by applying the even numbered free fatty acids from C_2 - C_{16} repeatedly under occlusive patch tests to human skin it was found that greater irritancy or penetration or both occurred with the C_8 - C_{14} range of fatty acids, especially with the C_{12} fatty acid. Again, R. E. Kellum, K. Strangfield and L. F. Ray,† studying

* *Arch. Derm.*, Vol. 97, No. 6, June, 1968, p. 722.

† *Ibid.*, Vol. 101, No. 1, January, 1970, p. 41.

the triglyceride hydrolysis by corynebacterium acneis (propionobacterium acneis) *in vitro*, were investigating along the lines of the hypothesis that free fatty acids irritate the sebaceous follicles and that in acne these free fatty acids are produced by the action of corynebacterium acneis breaking down the sebaceous material produced in the sebaceous glands. They used 63 strains of corynebacterium on three glycerides.

75 per cent. of *c. acneis* isolated from patients with acne vulgaris split C_{12} triglyceride (trilaurin).

56 per cent. hydrolysed C_{16} triglyceride (tripalmitin).

88 per cent. split C_{18} triglyceride (triolein).

The comparative figures for *c. acneis* strains from patients who did *not* suffer from acne vulgaris were:

42 per cent. split trilaurin;
17 per cent. hydrolysed tripalmitin;
58 per cent. split triolein.

The differences were significant and they feel that distinctive strains of *c. acneis* appear to inhabit the sebaceous follicle of the acne patient, strains with particular abilities to hydrolyse certain triglycerides; but in March, 1970, R. E. Kellum and K. Strangfield came to the conclusions that there were no qualitative differences between the median percentages and the ranges of the bacterial fatty acids from C_{10} - C_{18} found in isolates of *c. acneis* obtained from patients with and without acne. They believe that this excludes the theory that irritant fatty acids are derived from *c. acneis*.

I hope that I have been able to clarify the fact that acne is not only a teenage problem. It is a troublesome complaint amongst mature citizens and it can go on into old age. It would appear that there is still confusion in the theories of the finer details of the mechanism of acne; much remains to be confirmed re the hormonal processes, the mechanism of the keratin, the chemical processes

and the bacterial involvement; and perhaps when further advances have been made along these lines an explanation of why some patients suffer from acne and others do not, and why those who suffer from acne do so in varying degrees even within the same family, may come forth; and at the same time we may be able to arrive at a simple and effective treatment, the explanation of the use of which will not only be apparent to the medical practitioner, but will be easily explainable to the patients themselves.

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Table 1

CASES OF ACNE FOUND AMONGST EUROPEAN FEMALE PATIENTS OF A MATURE AGE

Case No.	Age	Marital state	Occupation	Colouring	Takes the pill	Took the pill	Children	Health	Previous acne	Duration of present acne	Pre-menstrual exacerbation	Related to confinement
1	40	M	Clerk	Red	No	No	Yes	Good	Nil	1 year	Nil	No
2	29	M	Housewife	Brunette	No	No	Yes	Fair	Nil	6 months	Nil	No
3	36	S	Clerk	Brunette	No	No	No	Good	Teenager	10 months	Yes	No
4	36	M	Housewife	Light brown	No	Yes	Yes	Good	?	6 months	Nil	No
5	34	M	Housewife	Brunette	No	3 mos.	Yes	Good	Nil	4 years	Yes	Yes
6	29	M	Farmer's wife	Brunette	No	No	Yes	Good	Yes	16 years	Nil	No
7	31	M	Housewife	Light brown	Yes	Yes	Yes	Good	Teenager	+ 9 months exacerb.	Nil	No
8	26	M	Housewife	Blond	No	No	Yes	Good	Nil	1½ years	Yes	No
9	27	D	Clerk	Blond	No	?	No	Good	Nil	3 months	Nil	No
10	27	M	Housewife	Brunette	No	No	Yes	Good	Teenager	1 year	Nil	No
11	32	M	Housewife	Brunette	?	?	Yes	Good	Teenager	1½ years	Nil	Yes
12	28	M	Housewife	Brunette	Yes	?	No	Good	Teenager	16 years	Nil	No
13	34	M	Housewife	Brunette	Yes	?	No	Good	Teenager	2 years	Nil	No
14	27	M	Housewife	Blond	?	?	Yes	Good	Teenager	12 years	Yes	No
15	34	M	Housewife	Brunette	Yes	?	Yes	Good	Nil	9 years	Nil	No
16	33	M	Housewife	Brunette	No	Yes	Yes	Good	Nil	4 years	Nil	Yes
17	31	M	Housewife	Blond	Yes	Yes	Yes	Good	Nil	9 years	Nil	Yes
18	39	M	Farmer's wife	Light brown	No	Yes	Yes	Good	Nil	2 years	Yes	Yes
19	32	M	Housewife	Blond	?	?	Yes	Fair	Yes	5 years	Nil	No
20	40	M	Telephonist	Brunette	No	Yes	Yes	Good	Nil	8 years	Nil	Yes
									Yes	28 years	Yes	No



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